4. Explanation of the Manifestations of Epilepsy. Boltzen.
5. Some Reflex Investigation, Namely, Concerning the Presence of Certain Reflexes. Würzen.

1. Pathological Anatomy of Acute Ascending Paralysis.—Some consideration is given to the three forms of myelitis as described by Schmaus, namely, parenchymatous degeneration, infiltration and softening. Other classifications are mentioned, particularly that of Lewandowsky. One case is reported and the individual came to autopsy; the histological findings are appended. The writer concludes that Landry's paralysis can through a degenerative process appear in a chronic intoxication. In the rapidly fatal case death may be due to bacterial toxines. The peripheral nerves are usually affected, but this is not absolutely necessary. The course of the disease may be so rapid that there is but slight evidence of acute morphological change.

2. Pathological Anatomy and Pathogenesis of Syringomyelia.—The material for this investigation was supplied by seven cases of syringomyelia, and in three of these the disease was combined with hydrocephalus. The article is elucidated by ten well selected illustrations and in these there is shown a widening of the central canal with a surrounding gliomatous proliferation. At times groups of glia cells are found so arranged as to present a glandular appearance and tumor-like areas of gliomatosis are likewise met with. Two cavities may be observed, one on either side of the cord and the appearance of a diverticulum may be presented.

The writer goes on to explain that through an excess of cerebrospinal fluid there is caused continuous pressure in the cavity and also irritation which leads to proliferation of the glial tissue. The epithelial layer becomes atrophied and may loosen and disappear. Through increased pressure by the fluid, the cord atrophies and the nerve fibers and cells degenerate. The clinical equivalent of the pathologico-anatomical changes are found in the triad of sensory, motor and trophic symptoms.

4. Explanation of the Symptoms of Epilepsy.—In writing upon this subject the author says that genuine epilepsy and numerous forms of cerebral are (in the immense majority of cases) cortical and cannot yet be distinguished; there is a similarity in the attacks and also in the secondary dementia. Cerebral epilepsy may occur after diseases of the meninges, the brain cortex or the deeper lying parts, which in general through a sclerotic process causes circulatory disturbance of the brain cortex. Genuine epilepsy is a chronic auto-intoxication arising through nutrition and metabolic disturbances, the consequence of hypofunction of the thyroid gland and epithelial bodies and the failure to eliminate sufficiently the poisons. In consequence of the hypothyroidism the elimination of many ferments and intermediate products is diminished. In cerebral as well as cortical epilepsy there is through the diminished circulation an accumulation of toxines in the brain cortex. The attack must be considered a reaction of the organism to free itself of the toxine. The blood gives its toxines off through the kidneys, lungs and skin, and the brain cortex can then give off its toxines to the toxine free blood. In
genuine epilepsy a rectal injection of the freshly expressed juice from glands corresponding to those showing an insufficiency may cause a subsidence of the symptoms.

In the cerebral forms of epilepsy a trephine operation may lead to a betterment of the condition.

6. Atrophic Myotony.—In this paper report is made of a man 43 years of age who showed how much the dystrophic and tabeticform symptoms may prevail in atrophic myotonia. Two illustrations demonstrate the facies myopathica and paresis of the orbicularis oculi. It is also brought prominently forward that trauma may be the inciting cause.

A careful search for vagotomy or sympatheticotomy did not reveal the presence of either bodily or pharmacological evidence of that condition.

Yawger (Philadelphia).

Monatsschrift für Psychiatrie und Neurologie

(Vol. 35, No. 1)

1. The Anterior Central Gyrus in Lesions of the Pyramidal Tracts and in Amyotrophic Lateral Sclerosis. P. Schroeder.
2. Feeblemindedness and Mental Affections with Dwarfism. W. Weygandt.
3. The Symptoms of Cerebellar Disease and their Significance. M. Rothmann.
5. The Question of Loss of Memory in Paralytics. M. Rohde.

1. Anterior Central Gyrus.—Several cases are described clinically and the autopsy findings are discussed. All cases of course showed destruction of the Betz cells and certain other large cells of the motor cortex as the most prominent feature. There was also a glia increase which did not correspond in location and probably not in time of development with the degeneration of the Betz cells. The six cases of pyramidal lesion tend to show further proof of the relationship between the pyramidal tracts and the anterior (not the posterior also) central gyrus. They do not show, however, that a direct and simple relationship of cell to fiber exists as in the case of the anterior horn cell and anterior root fiber. In fact there are certain observations which point to such a relationship not containing, e. g., the preservation of certain central fibers in the pyramid even when practically all the Betz cells are destroyed. Numerous photomicrographs accompany the article.

2. Dwarfism.—Attention is chiefly drawn to the multiplicity of causes. The author mentions no less than fourteen different etiological groups. All sorts of combinations occur. An interesting observation is that of a dwarf who again began to grow after the age of thirty years and reached a normal height but was poorly developed. Two similar cases are quoted from the literature.

3. Cerebellar Symptoms.—A didactic exposition of the symptomatology of cerebellar lesions. The article constitutes a valuable résumé and digest of the work done by all authors in this line to the present date. The cerebellar affections are susceptible of localization as to whether the lesion is in the cortex or nuclei, worm or hemisphere, just as in the cerebrum. Affections of the worm produce typical cerebellar gait, often with queer position of the head, speech is slow and indistinct. Lesions of the cortex of the cerebellar hemispheres cause symptoms of one side of the body or of one extremity. Ataxia and atonia occur in the same side as the lesion. Adiadochokinesis and loss of resistance reaction are usually present. The most marked symptoms are the variation and unnatural directions of the movements of the limbs in carrying out an act. Affections of the nuclei produce giddiness and dis-